CASE REPORT

Isolated optic nerve oedema as unusual presentation of electric injury

Saef Izzy,¹ Wissam Deeb,¹ George B Peters III,² Ann Mitchell¹¹Department of Neurology, University of Massachusetts Medical School, Worcester, Massachusetts, USA²Department of Ophthalmology, University of Massachusetts Medical School, Worcester, Massachusetts, USA

Correspondence to

Dr Saef Izzy, SIZZY@mgh.harvard.edu

Accepted 28 September 2014

SUMMARY

A 45-year-old man with no significant medical history presented following an electric current injury (380 V). He developed multiple systemic injuries including third degree burns and after 1 week of hospitalisation he reported unilateral visual changes. Examination suggested the presence of optic nerve oedema without evidence of haemorrhage, exudate or vessel abnormality. This was considered to be related to the electric shock. A trial of corticosteroids was considered. He was followed up to 5 months in clinic and was noted to have developed unilateral optic atrophy and no other systemic manifestations. Initial and 5 months follow-up optic nerve colour photograph and optical coherence topography were documented. The present case highlights the fact that electric current injury can present with only a unilateral ischaemic optic neuropathy, the need for early diagnosis for timely treatment and the controversial role of corticosteroids.

BACKGROUND

Ophthalmological manifestations are known to be one of the effects of lightning and electric injuries. The mechanism of neuro-ophthalmological injury is still not well understood with multiple mechanisms being suggested. These include the direct effect of electricity passing through ocular tissue, tissue ischaemia secondary to generalised vasoconstriction and in the case of cardiac dysfunction, cardiac arrhythmia causing retinal artery hypoperfusion.^{1–3}

It is also hypothesised that various mechanisms may lead to different types of damage.

Tissue susceptibility to injury varies. As the retina and optic nerve have lower electrical resistance, they are less susceptible to direct electric injury and more susceptible to indirect injury induced by vascular injury¹ (most likely secondary to induced thrombosis).

We report a case with a unique optic neuropathy caused by high voltage electric injury that resulted in optic nerve oedema associated with ipsilateral severe visual field constriction. To the best of our knowledge, the damage was not associated with any of the known or previously reported electric eye injuries: corneal changes, cataract, uveitis, keratitis, macular oedema and macular hole.^{3–7}

CASE PRESENTATION

A 45-year-old previously healthy man presented to the emergency department after being electrocuted by a 200–400 V AC electric power supply (switch-board) was electrocuted with his right arm catching

fire. On removing his arm from the electric box, he lacerated his wrist. The electricity travelled up his right hand, his right shoulder and exited his left knee. He suffered first-degree burns over the right side of his nose, right eye and he singed his facial hair. He also suffered second-degree burns on his right hand, right shoulder and left knee, there were also third-degree burns on his right hand and arm. In terms of his right wrist laceration, he lacerated ulnar nerve, ulnar artery, flexor carpi ulnaris (FCU) tendon and flexor digitorum superficialis (FDS) tendon. At the time of presentation, the patient complained of a sensation of a foreign body in his right eye. He was initially treated for presumed corneal abrasion. On hospital day 7, with decreasing sedation, he started to report peripheral vision loss in his right eye with photopsia and some central vision blurring. He was found to have a 3+ relative afferent pupillary defect on the right. His right eye had normal central near visual acuity (J1+) and 20/40 on the left. There was a dense generalised peripheral right visual field defect on confrontation and 2+ diffuse optic nerve oedema without haemorrhage, exudate or vessel abnormality. The anterior segments of both eyes and the posterior segment of the left eye were normal. The findings were consistent with optic neuropathy and were thought to be secondary to the electrical shock he had suffered.

INVESTIGATIONS

Serological investigations for other possible causes of optic neuropathy, including Lyme, syphilis and antineutrophil cytoplasmic antibodies were negative. A posteroanterior and lateral chest radiograph was normal. MRI of the brain and cervical spine were unremarkable. The patient had MRI orbits with and without gadolinium that was normal.

DIFFERENTIAL DIAGNOSIS

The patient's examination and clinical course suggest a form of non-arteritic anterior ischaemic optic neuropathy (NAION) due to electrocution. The differential diagnosis included infectious, inflammatory, vascular and demyelinating diseases.

TREATMENT

The patient was treated with 3 days of 1 g of intravenous methylprednisolone.

OUTCOME AND FOLLOW-UP

Two weeks later, the severe visual field constriction remained as well as 3+ right relative afferent pupillary defect. Distance acuity was 20/40 in each eye.

CrossMark

To cite: Izzy S, Deeb W, Peters GB, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2014-205016

Figure 1 (A) Initial right optic nerve colour photograph illustrating optic nerve oedema and slight pallor without associated haemorrhage. (B) Initial right optic nerve optical coherence topography analysis depicting nerve fibre layer enlargement about the superior and inferior temporal arcades.

Intraocular pressure was normal. Slit lamp examination was normal, including lens examination. Dilated examination remained unremarkable except for the continued 2+ diffuse right optic nerve oedema, without haemorrhage, exudate or

vascular abnormality (figure 1A). Optical coherence topography analysis was performed and depicted nerve fibre layer enlargement about the superior and inferior temporal arcades (figure 1B).

Figure 2 (A) Five months follow-up right optic nerve colour photograph illustrating significant optic nerve pallor. (B) Five months follow-up right optic nerve optical coherence topography analysis depicting generalised nerve fibre layer loss/atrophy.

Three months later (5 months following the injury), he still reported of blurred vision, a limited visual field and occasional, although less frequent, photopsia. Distance visual acuity improved to 20/20–2 (and remained 20/40 OS) with a continued relative afferent pupillary defect and severely restricted visual field on the right. Fundoscopic examination now revealed 2+ generalised optic disc pallor (figure 2A). Follow-up optical coherence topography analysis done and depicted generalised nerve fibre layer loss/atrophy (figure 2B). Neurologically, he was otherwise intact.

DISCUSSION

The clinical presentation resembles NAION.

Anterior ION is principally a group of disorders affecting the circulation of the anterior optic nerve, which is supplied by the short posterior ciliary arteries. AION has been classically divided into arteritic and non-arteritic.

Review of the literature finds only one recently reported similar case of optic nerve injury secondary to a lightning strike.³ However, in that case there were other ocular injuries accompanying the optic neuropathy.

In his paper 'Electric Injuries,' G. von Bahr included factors that would modulate the response to electric injuries: a minimal voltage of 200V; the rate of current flow; and the duration of the flow.⁷ The unilateral involvement in our patient could be explained by the supposed current trajectory. The limited pattern of injury might be related to the low energy of the current.

Our patient was treated with high dose methylprednisolone although this is still considered a controversial treatment option for non-arteritic anterior ischaemic optic neuropathy (NAION). Hayerh and Zimmerman in 2008 published a prospective cohort study investigating the role of systemic corticosteroids (80 mg prednisone daily with slow taper) in the acute phase of NAION on visual outcome. They reported statistically significant improvement of visual acuity and visual fields in patients treated with corticosteroids versus those not treated.⁸ In contrast a study published in March 2012 by Rebolleda *et al*⁹ that used the same steroid protocol, found no statistically significant difference in visual acuity between the two groups with more side effects and complications.

Certainly any efficacious treatment will need to be initiated in the early stages of NAION for maximum benefit. Currently no consensus exists about treatment of NAION or of patients with

electrocution-associated optic neuropathy, and further studies will be required to clarify this treatment issue.^{8–10}

Learning points

- ▶ Electric injury differentially effects organs and tissues. High clinical suspicion for isolated optic nerve injury should be considered as it might be the sole manifestation.
- ▶ The case highlights the clinical progression of optic nerve injury following electrocution that is reminiscent of non-arteritic anterior ischaemic optic neuropathy (NAION), though exact pathophysiology is not clear.
- ▶ No effective treatment has been established for management of NAION and further research is needed. The role of corticosteroids is controversial at best.

Contributors SI, WD, AM and GBP participated in the design/conception, writing the article and revising it.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Grover S, Goodwin J. Lightning and electric injury: neuro-ophthalmologic aspects. *Semin Neurol* 1995;15:335–41.
- 2 Frauntelder F, Meyer M, Andrews CJ, *et al*. Lightning injuries: Electrical, medical, and legal aspects. In: *Pathophysiology of lightning injury*. Boca Raton, FL: CRC Press, 1992;6:92–4.
- 3 Yi C, Liang Y, Jiexiong O, *et al*. Lightning-induced cataract and neuroretinopathy. *Retina* 2001;21:526–8.
- 4 Lagreze WD, Bomer TG, Aiello LP. Lightning induced ocular injury. *Arch Ophthalmol* 1995;113:1076–7.
- 5 Espallat A, Janigian R, To K. Cataracts, bilateral macular hole, and rhegmatogenous retinal detachment induced by lightning. *Am J Ophthalmol* 1999;127:216–17.
- 6 Jaffe GJ, Handa JT. Lightning maculopathy. *Retina* 1994;14:169–72.
- 7 von Bahr G. Electric injuries. *Ophthalmologica* 1969;1–3:109–17.
- 8 Hayreh SS, Zimmerman MB. Non-arteritic anterior ischemic optic neuropathy: role of systemic corticosteroid therapy. *Graefes Arch Clin Exp Ophthalmol* 2008;246:1029–46.
- 9 Rebolleda G, Pérez-López M, Casas-Llera P, *et al*. Visual and anatomical outcomes of non-arteritic anterior ischemic optic neuropathy with high dose systemic corticosteroids. *Graefes Arch Clin Exp Ophthalmol* 2013;251:255–60.
- 10 Atkins EJ, Bruce BB, Newman NJ, *et al*. Treatment of nonarteritic anterior ischemic optic neuropathy. *Surv Ophthalmol* 2010;55:47–63.

Copyright 2014 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow